

## 9

# Microcurrent therapy in the treatment of fibromyalgia

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## Carolyn McMakin

The author has been using microamperage current modulated by specific frequencies to treat both myo-fascial pain and fibromyalgia since 1996. This new treatment technique makes these conditions much more responsive to treatment and has allowed some patients to recover. In this context, recovery means the patient no longer meets the diagnostic criteria for fibromyalgia, sleeping well without medication and having less than 11/18 tender points when tested using the American College of Rheumatology (ACR) protocol. The patient might still need treatment for specific joint or muscle problems that would be normal for someone else of the same age but the overall fibromyalgia complaints of fatigue, sleep disturbance and generalized aching are gone, often permanently. This chapter will present the history and theory behind frequency specific micro-current and describe the treatment techniques that allow us to treat myofascial pain, neuropathic pain and fibromyalgia with some measure of success.

## MICROCURRENT

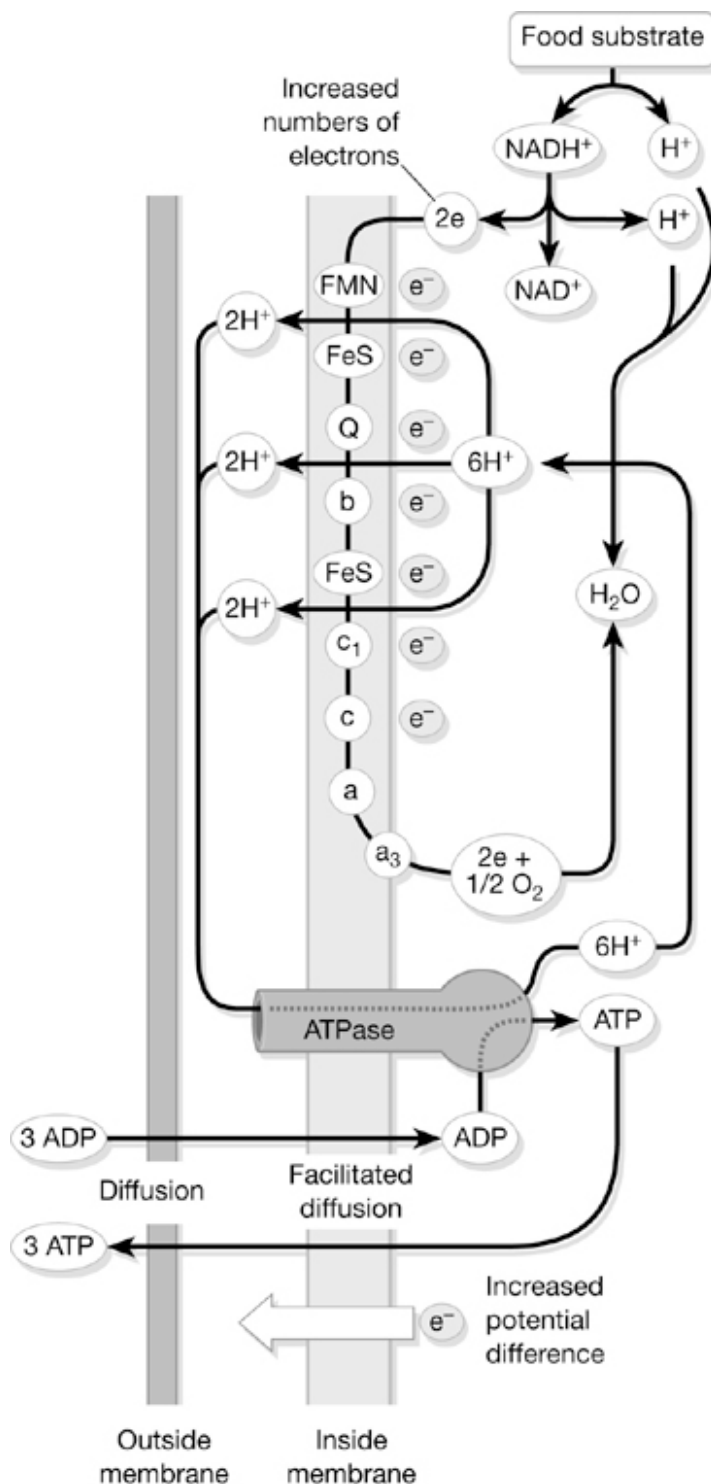
Microamperage current was introduced in the USA in the early 1970s (Rowley et al 1974). Microcurrent provides current to the patient in a physiologic range of microamperes or millionths of amperes. An ampere (amp) is a measure of the strength of electric current and measures the rate of flow of charge in a conducting medium. One micro amp (mA) equals 1/1000th of a milliamp (mA). By comparison, interferential, TENS, and high-volt pulsed galvanic stimulators deliver currents in the milliamp range causing muscle contraction, pulsing and tingling sensations. With microcurrent the patient cannot feel the current since there is not enough current to stimulate sensory nerve fibres (Mercola & Kirsch 1995).

Microcurrent has typically been used to increase the rate of healing in injured athletes, and to control pain, increase the rate of fracture repair and treat myofascial pain and dysfunction (Manley 1994, Morgareidge & Chipman, 1990 Rowley et al 1974).

In a study conducted in rat skin by Ngok Cheng MD, electrostimulation of the tissues with microcurrent resulted in remarkably increased ATP concentrations, protein synthesis and membrane transport. With currents from 50mA to 100mA, the ATP levels were increased threefold to fivefold. With currents from 100–500mA, the stimulatory effects were similar. With currents exceeding 1000mA the ATP concentration levelled, and with 5000mA they were even reduced slightly as compared with the non-treated controls. Similar effects were noted in regard to protein synthesis. At about 500mA there is a tremendous enhancement of protein synthesis, but when the current rose over 5000mA the trend reversed into suppression (Cheng 1982).

Normal membrane bioelectric activity includes the flow of electrons through the electron cascade in the cell wall to produce ATP. The first step in oxidative phosphorylation in the mitochondria involves the ionization of the hydrogen atoms that have been removed from the food substrates. The hydrogen atoms are removed in pairs; one immediately becomes H<sup>+</sup> and one combines with NAD<sup>+</sup> to form NADH. The electrons that are removed from the hydrogen atoms to cause ionization immediately enter the electron transport chain in the inner mitochondrial membrane. The transport of these electrons through the electron transport chain releases energy that is used to synthesize ATP. For each two electrons that pass through the entire electron transport chain, up to three ATP molecules are synthesized. The large electrical potential difference between the inner and outer mitochondrial

membrane causes the hydrogen ions to flow into the mitochondrial matrix through the ATP-ase molecule. The greater the electron flow, the greater this electrical potential difference and the greater the creation of ATP. Microcurrent could increase the production of ATP by both of these mechanisms (Guyton 1986) (Fig. 9.1).



**Figure 9.1** The illustration shows membrane electron transport and two possible mechanisms by which microcurrent could produce the 500% increase in ATP production described by Cheng in 1987. One mechanism involves the increase in the number of electrons provided by the microcurrent flowing across the membrane through the electron transport chain. The other mechanism involves an increase in the electrical potential difference across the membrane created by the electrons provided by the microcurrent. The greater the potential difference, the greater the production of ATP. Both mechanisms may contribute to the observed effect. (Adapted from Guyton 1986.)

The author uses a standard battery operated microcurrent instrument made by Precision Microcurrent Inc (Fig. 9.2). This has two channels that can each be set to a different frequency from 0.1Hz to 999Hz. Previous models had two-digit specificity with a three place multiplier, so the digits 2 and 8 could become 2.8HZ, 28Hz or 280Hz depending on the multiplier. The frequencies used require three-digit specificity and in current models the frequencies are accurate to three places. Thus the frequency 284Hz can be set with less than 1% error in output and can become

28.4 by changing the multiplier. Each channel generates current in a ramped square wave form from 10 to 600 micro amps. The circuitry modifies the voltage so that the current remains constant regardless of tissue resistance. The circuitry also modifies the DC battery current so that it can alternate or be polarized. This feature is important because most treatments are carried out using alternating current but in the author's experience nerve tissue is most effectively treated with current polarized positive centrally and negative distally. Any microamperage current generator could be used as long as it has these characteristics.



**Figure 9.2** The photograph shows the battery operated, two-channel microcurrent instrument used in treatment (manufactured by Precision Microcurrent). The controls (from left to right along the upper portion of the instrument) are: the buttons to set frequencies for channel A, the threshold adjustment (not used in the treatments described in this chapter), the switch that changes the polarity from alternating (for myofascial treatments) to positive (for nerve and spinal cord treatments) and, last, on the right, the buttons to set the frequencies for channel B. The controls in the lower row (from left to right) are: to select the current for channel A, the current for channel B, to set the timer for either continuous or timed segments, and to select the wave slope from sharp (for sedating nerves) to gentle (for treating new injuries and fresh wounds). LED lights above each current selection knob indicate the activity of each channel. The meter on the right shows what percentage of the current output is reaching the patient. There are two leads for each channel colour coded red and black for positive and negative polarity.

## Using specific frequencies

The early microcurrent instruments provided current modulated by only a few frequencies: 0.3Hz was used to stimulate healing, 30Hz or 40Hz were found useful to reduce pain, and 300Hz was used clinically for lymphedema (Greenlee & Wing 1986, Manley Tehan 1994). The frequencies employed by the author were those used by a retired osteopathic physician who found them in a manual that came with a 1920s electromagnetic therapy device he acquired when he bought a practice in Canada. Using this ancient machine he used one frequency to address a particular tissue or organ and a second frequency to neutralize a pathology or support a function in that tissue, and had a reputation for helping people to recover from various difficult conditions. When he died and the machine became defunct, two Oregon chiropractors, who had trained with him, saved the frequencies for historical interest. In 1994 one of these chiropractors provided the frequencies and guided the author in using Van Gelder's treatment protocols on a two-channel microcurrent instrument. Later, frequencies were added taken from the work of Albert Abrams, a medical physician who practised in San Francisco in the early twentieth century (Abrams 1934). Several additional frequencies were developed through trial and error.

Of all the millions of permutations and combinations of frequencies that could possibly have an effect on biologic tissue, why do these particular frequencies seem to work in a microcurrent system? We cannot know with certainty how the original practitioners arrived at these frequencies. In clinical applications certain frequencies either work or they do not work (and if they do not work they have no obvious negative effects). As a result, clinical outcomes and treatment response have been the criteria as the determination of efficacy. As with the use of willow bark and then

aspirin for hundreds of years before the chemistry of prostaglandin inhibition was understood, medicine does not always have to understand the mechanism and the precise origin of the therapy to make use of it.

The frequencies are rarely used as a single frequency. A process of trial and error has shown that using one frequency thought to address or neutralize a condition on channel A and one thought to be targeting a specific tissue on channel B produces an optimal effect.

The combination of the two frequencies seems to be important since changing either channel A or channel B changes the clinical response. The frequencies are applied in an interferential pattern so that they intersect in the area to be treated. Because of the interferential effect a complex of frequencies is being created in the treatment field that includes each frequency by itself, the sum of the two frequencies and their difference. The microcurrent instrument creates a ramped square wave with a 2.5-second pulse to carry the frequency. Square waves are produced by high frequency spikes that rise and drop off sharply. At present there is no way of knowing what portion of this complex is necessary to create the observed therapeutic effects but it is clear in a clinical setting that changes in either the frequency or the wave shape alter the therapeutic effect.

## History and theoretical model for specific frequencies

The microcurrent instrument acquired by the author in 1994 was the first one available that had the ability to provide a specific frequency on each of two channels. In 1996 a different microcurrent instrument was obtained. This was designed to be used in cosmetology for skin treatments and came with a pair of graphite gloves designed to be worn by the practitioner to conduct the current from the instrument to the patient (Fig.9.3). These lightweight gloves have micro-jacks cemented to the dorsal surface and are designed to conduct current and provide good tactile perception. The gloves seemed ideal to conduct current into injured muscles and in January 1996 the gloves, the two-channel microcurrent instrument and the frequencies to neutralize 'mineral deposits' were combined in order to treat a patient with resistant myofascial pain. The fibrotic tender tissue softened within seconds and became pain free in minutes after being unresponsive to weeks of manual trigger point therapy.



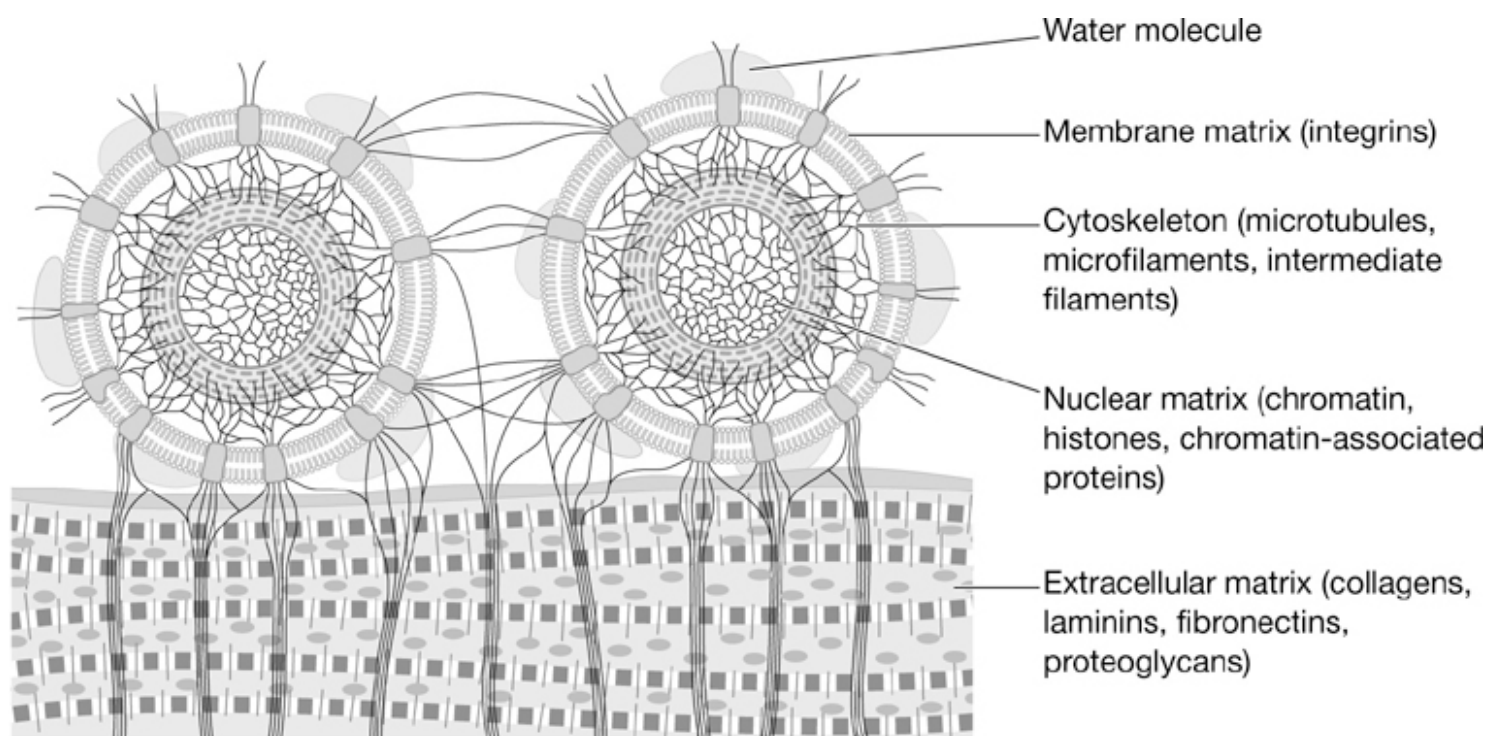
**Figure 9.3** The lightweight graphite conducting gloves have both red leads connected to one glove and both black leads attached to the other glove through double pin-jacks cemented on the back of the glove.

The author began experimenting with the treatment of chronic myofascial pain in 1996. After the first 150 patients it was clear that the results were both consistent and very frequency specific (McMakin 1998). Patients responded and recovered who had been symptomatic for years despite skilled and appropriate treatment from a variety of practitioners. When certain frequencies were applied the tissue would soften dramatically in seconds, accompanied by reduction in pain. Not all frequencies nor all frequency combinations produced this response. An 'inappropriate' or ineffective frequency produces no change in tissue no matter how long the frequency is used and changing to a 'correct' frequency produces the characteristic softening of the tissue in seconds.



How could specific frequencies produce specific effects in different tissues in the ways observed? Biophysics provided an intellectually satisfying foundation for the observed effects. The explanation for the effects of specific frequencies on specific tissues and conditions starts with a quantum view of physical tissue instead of a Newtonian or mechanical view. Physical tissue looks solid but, in reality, it is a collection of biochemicals formed, folded and aligned in particular configurations to create a biological/biochemical/bioelectric system. The molecules and atoms that create these biochemicals are held together by electromagnetic bonds in an energetic relationship. In fact, the atomic and subatomic particles that form the atoms are not matter at all but rather bits of energy that may behave as particles or as waves. There is more space between the particles than there are particles and the simple laws of physics dictate that the particles, atoms and molecules create an electromagnetic field in this space. This field must then be able to be influenced by other electromagnetic fields and may then have an effect on the tissue that creates it (Oschman, 1994, 1997, 2000).

On a macromolecular level the cell is no longer seen to be an unstructured bag of membranes filled with organelles processing reactions through simple diffusion. All of the intracellular organelles are suspended and interconnected by the microtrabecular lattice that forms the ground substance within the cell. Glycoproteins extend across the cell surface from the interior to the exterior and create a filamentous tissue network. This network is a crystalline gel lined by water molecules and functions like a semiconductor. Szent-Gyorgyi suggested that virtually all of the molecules forming the living matrix are semiconductors: 'Molecules do not have to touch each other to interact. Energy can flow through the electromagnetic field ... The electromagnetic field, along with water, forms the matrix of life. Water can form structures that transmit energy' (Oschman 1997) (Fig. 9.4).



**Figure 9.4** This figure shows the membrane structure that allows tissue to act as a semiconductor. (Reproduced with permission from Oschman 1997.)

This semiconductor tissue conveys and stores current, charge and vibrational information (Oschman 1997). The concept of a continuum between the brain and the rest of the body through the perineurium or fascia combined with the ability of tissues to hold energetic patterns would explain the body information is 'stored' in physical tissue and affects physical function. It would explain, for example, how the effects of physical injury, emotional trauma or toxicity exposure can remain in the tissue long after the tissue should have healed.

It also explains how electromagnetic patterns applied as frequencies could resonate with specific tissues and neutralize the patterns created by different conditions.

This method of treatment was first taught in 1997 to see if students could reproduce the results and by 1998 it was clear that the results were indeed reproducible. The results of microcurrent treatment of 73 cases of cervical and low back pain were presented at the American Back Society in 1997 and 50 cases of resistant myofascial pain in the head, neck and face were published in 1998 (McMakin 1998). Since that time thousands of cases of traumatic injury, chronic myofascial pain and nerve pain have been treated by trained microcurrent practitioners with similar results. In 1999 treatment protocols were developed that allowed successful treatment of neuropathic pain and the full body pain from fibromyalgia associated with cervical trauma (see below).

Frequencies and treatment protocols have been established by observing clinical response and have been refined by clinical experience and practitioner and patient feedback. Teaching consists of an 20-hour introductory course

and a 12 hour advanced course for physicians and therapists licensed to use electrotherapies. Trained practitioners can achieve certification in the USA and Australia after 6 months practise, the submission of 10 written cases, and passing a written examination. The method is so new that no controlled trials have been done as yet but they are planned as more research-oriented physicians become interested because of the clinical outcomes.

## TREATING FIBROMYALGIA ASSOCIATED WITH CERVICAL TRAUMA

In the author's fibromyalgia clinic it has been observed that fibromyalgia patients seem to divide into seven fairly clear though often overlapping aetiologies (these are clinical distinctions based on patient history and response to treatment developed during 6 years of treating hundreds of fibromyalgia and myofascial pain patients):

1. *Stress*: One type of fibromyalgia seems to be associated with prolonged emotional or physical stress and the subsequent adrenal depletion and physiologic sequelae of prolonged elevated cortisol and other stress hormones.
2. *IgG allergies*: One type is associated with 'leaky gut' and IgG food or environmental allergies. Mast cell overload from elevated IgG complexes causes the release of histamine, which stimulates class C pain fibres.
3. *Toxicity*: Another is associated with one time acute or long-term chronic exposure to organic chemicals, heavy metals or pesticides.
4. *Genetic*: One type has a genetic link, seems to run in families and may be associated with food sensitivities, especially gluten, or may represent an increased need for enzyme substrate in the liver detoxification pathways or serotonin pathways.
5. *Viral*: One type occurs after immunizations or viral illness.
6. *Vestibular injuries*: One type is associated with the sleep disturbances and cognitive difficulties resulting from a vestibular injury or brain trauma.
7. *Cervical trauma*: And there is one type of fibromyalgia that occurs after whiplash injuries, cervical trauma, or after surgery. The post-surgical cases are thought to occur when the neck is hyper-extended during intubation and constitute a cervical injury.

These different types have the same neuroendocrine and central sensitization features described in the fibromyalgia research but they each respond to different treatment strategies (Bennett 1999, Crawford 1998, Neeck & Reidel 1999). The model for these aetiologies has come from successful resolution of fibromyalgia when the treatment strategies address the specific cause of the pain or dysfunction.

It is the author's contention that fibromyalgia associated with cervical trauma (cervical trauma fibromyalgia or CTF) represents a distinct aetiology from fibromyalgia associated with other causes. The fully referenced theoretical model for the causative link between cervical trauma and fibromyalgia is presented in [Box 9.1](#). In summary, the author believes that cervical trauma cracks the disc annulus and exposes the spinal cord to the nucleus pulposus and neurotoxic concentrations of phospholipase A<sub>2</sub> (PLA<sub>2</sub>). PLA<sub>2</sub> is known to reduce the firing of nerves and may damage the anterolateral pathways, which are directly adjacent to the portion of the disc found to be injured in trauma. If the function of the anterolateral pathways was sufficiently slowed it could constitute a chemical lesion, or functional deafferentation, in the nociceptive system creating what is essentially central or thalamic pain. The pain descriptors for central pain and its affective quality are strikingly similar to the descriptors and affective quality seen with CTF. This hypothesis was developed after a literature search when it was found that the pain could be eliminated by treating the spinal cord.

### Box 9.1 Full hypothesis of the link between cervical trauma and fibromyalgia

We know that cervical trauma causes cracks in the annulus and fractures in the endplates that expose the nucleus pulposus to the spinal fluid either directly or via the spinal vasculature. In their paper on disc injuries in cervical trauma, Taylor & Twomey (1993) described the pathological changes in the cervical discs created by trauma. They did a comparative study of the cervical spines from 16 subjects who died of major trauma and 16 subjects who died of natural causes. Fifteen of the 16 trauma subjects showed clefts in the cartilaginous endplates. The cartilaginous endplates are important because they are vascularized whereas the disc itself is avascular. The vasculature extends from the endplate into the epidural space (Netter 1991). Age-related changes in the discs are found extending from the uncovertebral joints medially towards the centre of the disc and do not involve the endplates. Posterior disc herniations and facet haemarthrosis were also observed in the trauma group and absent in the control group (Taylor & Twomey 1993).

The outer annulus of cervical discs is innervated and cervical discs may be more extensively innervated than lumbar discs (Bogduk et al 1988, Mendel & Wink 1989). ... The rim lesions described by Taylor and Twomey usually involve the outer part of the annulus. The delayed healing and predisposition to premature degeneration after the experimental production of rim lesions in the intervertebral discs of sheep, with the tendency to vascularization of these lesions, suggest that something similar may be responsible for chronic pain associated with soft-tissue injuries to the

cervical spine. This study and the study of Davis et al (1991) show that neck extension sprain, with posterior disc herniation, may be associated with injury to the spinal cord such as localized petechial hemorrhage in the anterior columns or vascular damage to the anterior spinal or radicular arteries.

In the cases studied by Taylor & Twomey X-rays taken before microscopic examination did not show fractures, dislocations or subluxations: 'Clinical studies show that rim lesions and traumatic herniations are demonstrable in survivors of motor vehicle trauma in the absence of vertebral fractures.'

Patients with chronic residual cervical pain following trauma have symptoms that persist for years after soft tissue injuries should have healed. The damage to the cartilaginous endplates and discs provides a model for that persistence. Rim lesions produced surgically to a depth of 5mm in the discs of sheep do not heal when followed for a period of 18 months. Only the outer third of the annulus shows the capacity to heal. These disc and rim lesions extend inward to at least that depth with deformation of the annular lamellae and degeneration of the nucleus pulposus. This phenomenon helps to explain the persistence of the injury. (Taylor & Twomey 1993).

## NUCLEUS PULPOSUS AS A NEUROTOXIC AGENT

It has been established that the nucleus pulposus causes an inflammatory response in nerve tissue (Olmarker et al 1995). Nucleus pulposus material was implanted in the epidural space in pigs near the cauda equina. Implantation of retroperitoneal fat was used as a control. Nerve fibre degeneration, axonal swelling, increased axoplasmic density and marked attenuation and splitting of the myelin sheaths were noted in the animals exposed to nucleus pulposus material. Nerve root conduction velocity was significantly lower in the nucleus pulposus exposed nerve roots than in the control nerve roots. This study was the first time it had been demonstrated that the nucleus pulposus could produce reduction in nerve conduction velocity and nerve fibre degeneration without a mechanical compression of the nerve root (Olmarker et al 1993).

Nerve conduction velocity	Day 1	Day 3	Day 7
Control	84+/-2	83+/-4	76+/-11
Nucleus pulposus	63+/-9	45+/-16	45+/-19

It is known that the nucleus pulposus elicits an inflammatory response as indicated by leukotaxis and an increase in vascular permeability. The exact mechanism of the inflammatory response has not been verified. It is not clear whether the inflammatory reaction is induced by the nucleus itself or from substances being liberated from other tissues as a response to the interaction with the components of the nucleus. Glycoproteins, immunoglobulin G, phospholipase A<sub>2</sub> and hydrogen ions have been proposed as possible mediators of this inflammatory damage (Olmarker et al 1993).

Marshall proposed a 'chemical radiculitis' in which the annulus fibrosis is weakened by degeneration and finally ruptures under the stress of a traumatic episode. The nuclear fluid, which may be highly irritating to nerve tissue, is then ejected into the peridiscal tissue (Marshall et al 1977). The inflammatory properties of the nucleus pulposus have been demonstrated in hogs (Olmarker et al 1995), dogs (McCarron et al 1987) and rabbits (Cavanaugh et al 1997). Macrophages predominate in an area of tissue injury within a few days and secrete the by-products of phagocytosis, including hydrogen peroxide, lactic acid and PLA<sub>2</sub>. PLA<sub>2</sub> is an important lipolytic enzyme in the arachidonic acid cascade. This process intensifies and prolongs the inflammatory responses. Proinflammatory substances such as interleukin-1 and other cytokines may also activate PLA<sub>2</sub> and other proteolytic enzymes that are found in disc tissue. Phospholipase A<sub>2</sub> is present in high concentrations in herniated and painful discs (Ozaktay et al 1998).

It is concluded that immediate neural response is a direct effect of PLA<sub>2</sub>, based on its chemical composition. ... In a long term PLA<sub>2</sub> study 3 days after the application [of PLA<sub>2</sub>] breakdown of myelin sheaths, unclear axonal margins and vacuolar degeneration were observed ... The PLA<sub>2</sub> found in the herniated human disc may be neurotoxic around the immediate exposed tissues ... The evidence for neurotoxicity included loss of spontaneous nerve discharge after PLA<sub>2</sub> application and absence of response to mechanical stimulation in previously responsive units. (Ozaktay et al 1995)

Central processing of pain may arise from the neurotoxicity or recruitment of silent units. Long-term consequences of this neurotoxicity could include neurodegeneration, neural regeneration, neuroma formation and ectopic nerve impulses all of which can be sources of pain (Bennett 1994, Chen et al 1997). Peripheral nerve lesions can produce spinal cord changes that may contribute to deafferentation pain. These changes include sprouting of myelinated fibers into lamina 2 of the spinal cord and increased discharge of dorsal horn neurons. (Marshall et al 1977, Ozaktay et al 1998)

## FIBROMYALGIA AND PAIN PROCESSING

Robert Bennett (1999) has described the central sensitization of pain perception in fibromyalgia patients in wonderful detail. Pro-inflammatory cytokines (interleukin-1 and 6 and tumour necrosis factor) sensitize second order dorsal horn neurons (lamina V slow C multimodal pain neurons) through an NMDA–substance P–nitric oxide cascade. Mountz et al (1995) used SPECT scanning to demonstrate reduced blood flow to the thalamus and caudate nucleus where pain stimuli are processed in fibromyalgia patients as compared to normal controls. In acute pain, blood flow is increased in these areas. Bennett also points out that lesions of the lateral thalamus often result in a pain syndrome characterized by affective distress, and aching, burning and tingling pain that is exacerbated by normally innocuous stimuli such as light touch. This phenomenon is known as allodynia.

## CHRONIC PAIN AND CERVICAL TRAUMA

All of these pieces come together in the spinal cords of patients who have had cervical trauma. We hypothesize that the exposure of the nucleus pulposus material to the spinal fluid via the cracks in the annulus created by cervical trauma causes an inflammatory response in the spinal fluid. This inflammatory response may be mediated by phospholipase A<sub>2</sub> and its associated cytokines.

Nerve destruction such as that shown in dogs, pigs and rabbits would be created by these neurotoxic inflammatory chemicals in the spinal cord in response to exposure to the nucleus pulposus. Phospholipase A<sub>2</sub> has been shown to be so neurotoxic that it is capable of damaging the pathways in the anterolateral system. This inflammatory response is dose related (Ozaktay et al 1995, 1998). We hypothesize that this nerve destruction creates a chemical lesion in the paleospinothalamic tracts. These tracts carry pain information up the spine to the thalamus, caudate nucleus and the cortex and ascend the cord in the anterolateral portion of the lateral column. The paleospinothalamic tract is the outermost of the two tracts and carries diffuse deep chronic pain sensation (Bennett 1994, Netter 1991).

The anatomic proximity of the cracks in the discs and endplates could expose this system to high levels of these inflammatory chemicals. This tract is immediately adjacent to the site of the disc herniations demonstrated by Taylor & Twomey in their study of cervical trauma cases (Kandel & Schwartz 1985, Taylor & Twomey 1993).

The inflammatory damage to the anterolateral columns could operate in one of two ways. If the damage was minor and simply reduced the firing threshold of the axons, pain traffic up the cord would be facilitated and enhanced creating the profuse allodynia seen in fibromyalgia patients. If the inflammatory damage progressed to nerve destruction of the paleospinothalamic nerves, it would effectively create a chemical deafferentation. We have seen how inflammatory chemicals reduce the firing of the nerves and slow nerve conduction. The trauma induced physical damage to the anterolateral pathways and the cord seen by Taylor & Twomey add another possible aetiology for the deafferentation phenomenon. Deafferentation, whether caused by chemical disruption or physical trauma and damage in the ascending pain pathways, would produce what is essentially a thalamic pain pattern. I find this mechanism the more likely of the two proposed.

In their chapter on pain Kandel & Schwartz (1985) state that:

Central pain can arise not only from pathologic lesions in the thalamus but also from neurosurgical lesions placed anywhere along the nociceptive pathway from the spinal cord and brain stem to the thalamus and cortex. ... The sensations are unpleasant and abnormal, often unlike anything the patients had ever felt before: spontaneous aching and shooting pain, numbness, cold, heaviness, burning and other unsettling sensations that even the most articulate patients find difficult to describe. Central pain is particularly distressing emotionally.

## FIBROMYALGIA AND CENTRAL PAIN

Fibromyalgia patients with a cervical trauma aetiology have been describing this type of pain to me for 4 years. The description of the pain sensations associated with central or thalamic pain is precisely, word for word, what has been described in patient histories in more than 40 of our patients.

Deafferentation in the anterolateral pathways is capable of creating the tract lesions that produce the thalamic pain symptoms we see in fibromyalgia patients. Patients with fibromyalgia not caused by cervical trauma do not have the same quality of pain that cervical trauma patients describe. Their pain is diffuse and aching but it lacks the disturbing affective neuropathic intensity seen in the cervical-trauma mediated fibromyalgia patients. This affective intensity is characteristic of thalamic or central pain. The similarities between centrally mediated pain and the pain described by this group of fibromyalgia patients, and the differences between treatments effective in this group of fibromyalgia patients and other types of fibromyalgia patients, led to the development of this hypothesis.

The clinical picture suggests that the chronic central nerve pain facilitates the sympathetic nervous system



causing a chronic fight or flight response, especially when the disc is damaged at the C5–6 level causing stimulation/facilitation of the C5 sympathetic ganglion. The sympathetic response is characteristic. The body's repair systems are put on hold, circulation to the digestive system is reduced, myofascial circulation is altered, immune system function is compromised, the adrenals produce elevated levels of endogenous cortisol and are constantly taxed to keep up and the system gradually experiences more and more dysfunction. When the gut is compromised in this fashion for a year or more it is more prone to dysfunction, including 'leaky gut' and the resultant food and systemic allergy reactions. Elevated endogenous cortisol levels cause thinning of the gut wall and may impair transport of the branch chain amino acids. The branch chain amino acids are necessary precursors of neurotransmitters including serotonin, adrenaline (epinephrine), noradrenaline (norepinephrine), oxytocin and dopamine and are essential co-factors in phase one and phase two liver detoxification pathway function. Branch chain amino acid levels are reduced in fibromyalgia patients (Juhl 1998).

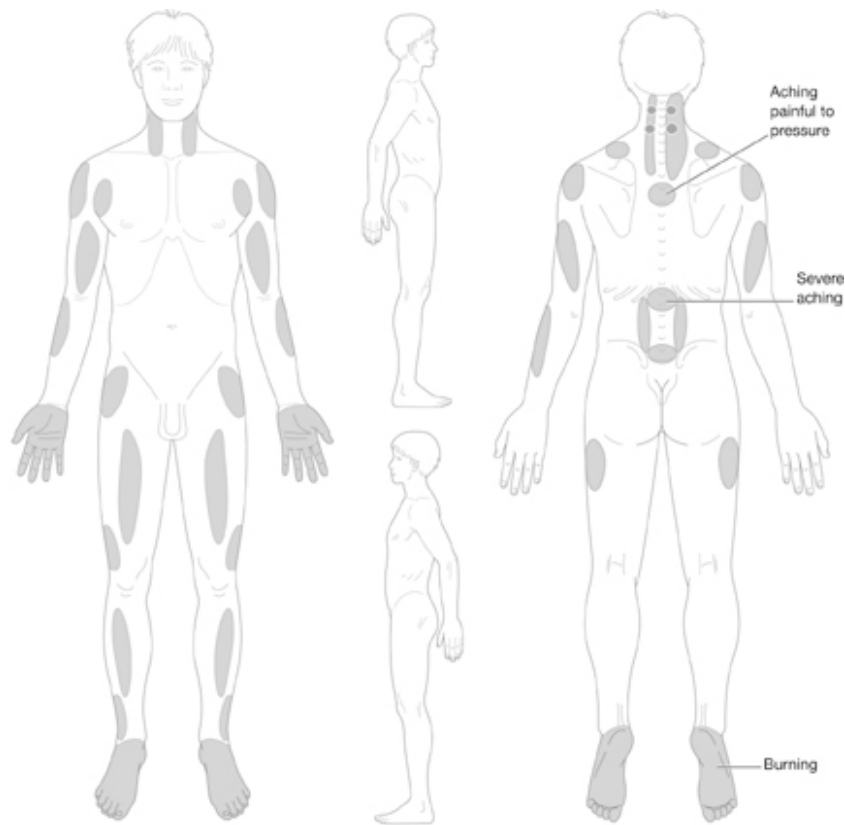
Adrenal fatigue follows inevitably after years of sympathetic upregulation and increased adrenal demand. By the time the patient has been in this condition for 1–2 years the symptoms have generalized into the classic neuroendocrine chaos we call 'fibromyalgia'.

This would all be interesting as an academic exercise but it becomes compelling when one is able to treat and reverse these effects.

In the author's experience, patients with fibromyalgia associated with cervical trauma localize their pain in the neck, arms, hands, midscapular and paraspinal area, gluteals, legs and feet. As a group, they are the only fibromyalgia patients who describe burning or aching pain in the hands and feet. The pain is usually rated subjectively on a visual analogue scale (VAS) between a 6 and a 9/10. Cervical trauma fibromyalgia patients use different pain descriptors than other fibromyalgia patients. They use words like 'burning', 'stabbing', 'sharp' and 'shooting' to describe their pain rather than the dull diffuse aching described by other fibromyalgia patients (Fig. 9.5). In general, they tend to have a higher incidence and greater severity of headaches and there is a characteristic affective quality to the pain that is reminiscent of central pain (Kandel & Schwartz 1985). The pain is not only moderate but also emotionally bothersome and irritating and is quite different from the pain described in fibromyalgia not associated with cervical trauma.

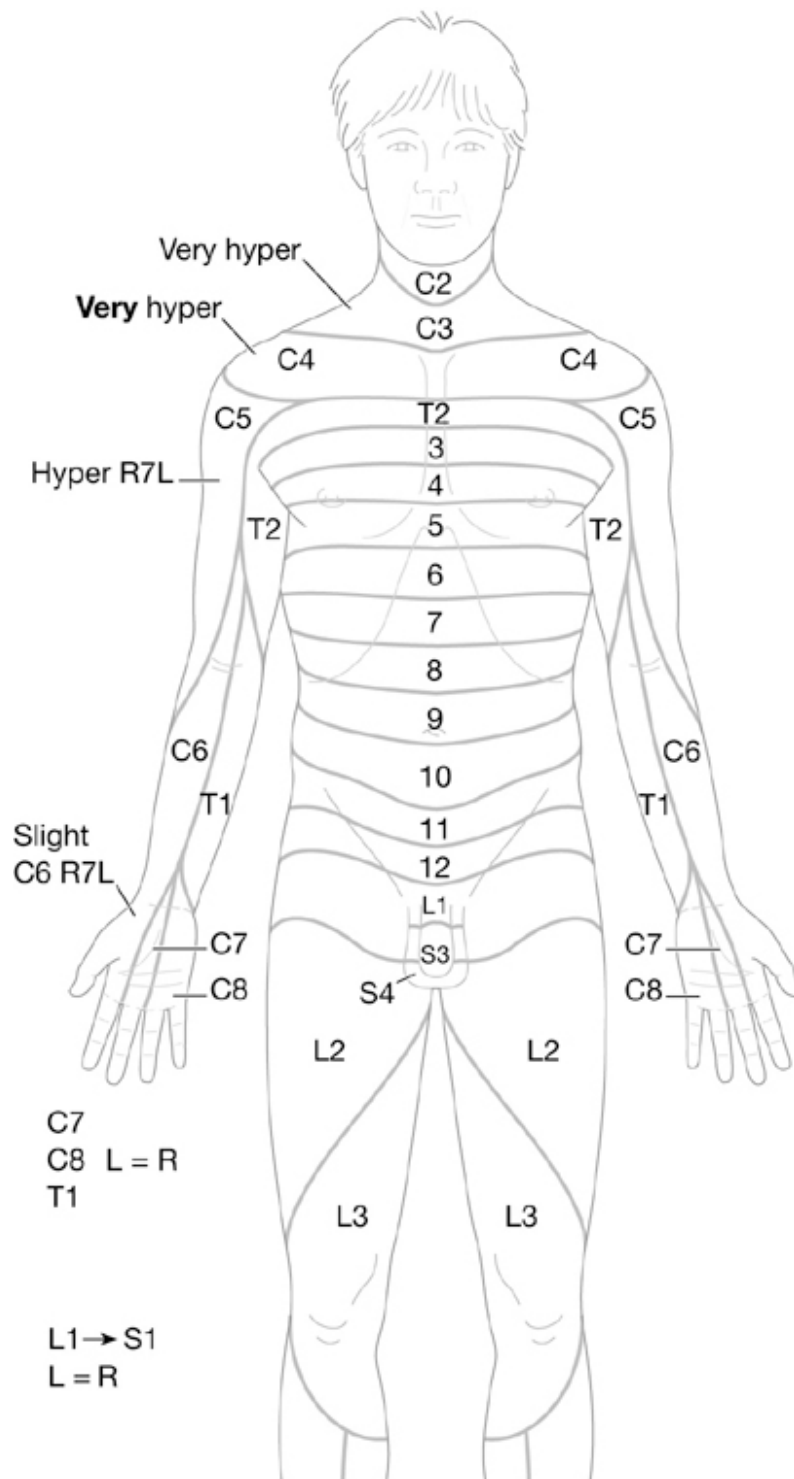
The neurologic examination findings for patients whose fibromyalgia is associated with cervical trauma are also different from fibromyalgia patients with a non-traumatic onset. Every trauma-onset patient had slight to moderate hyperreflexia of the patellar reflexes, many with cross reflexing, and some also had hyper-active triceps, biceps or abdominal reflexes. The sensory examination with Wartenberg's pinwheel showed dermatomal hyperaesthesia, usually at the C3, C4, C5 and C6 dermatomes (Fig. 9.6). This hyperaesthesia was distinct from and in addition to the allodynia characteristic of fibromyalgia regardless of aetiology. The neurologic examination suggests a degree of cervical cord and nerve irritability not seen in other types of fibromyalgia.

In any case, based on the physical examination findings CTF is clearly neuropathic pain somehow associated with cervical cord irritability. Chronic moderate pain would cause the elevations in corticotropin-releasing hormone (CRH) found in fibromyalgia and the alterations in central pain processing common to fibromyalgia patients (Bennett 1999). By the time the patient has been in this condition for 1–2 years the symptoms have generalized into the classic neuroendocrine abnormalities seen in fibromyalgia of any aetiology (Crawford 1998, Neeck & Reidel 1999.) This model was developed after it was found that we could successfully treat the pain associated with CTF by treating the spinal cord.



**Figure 9.5** Pain diagram typical of cervical trauma-induced fibromyalgia. As a group these are the only fibromyalgia patients to describe burning or aching pain in the hands and feet.

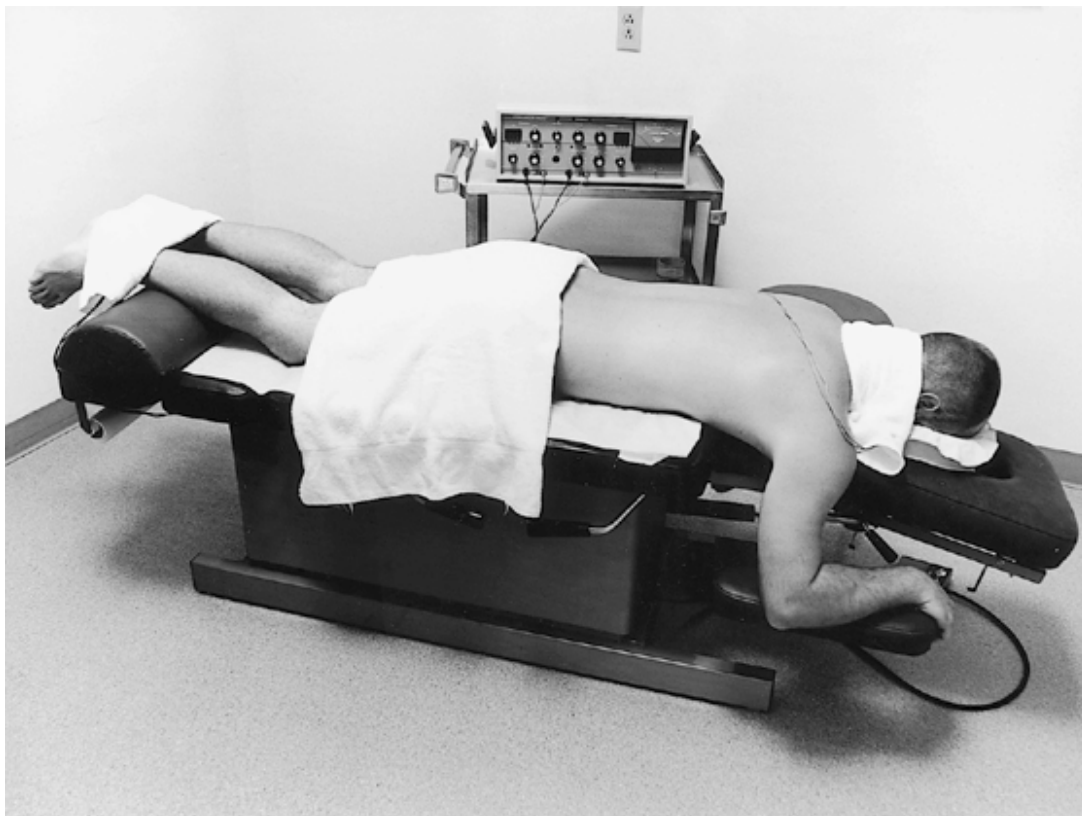
In February 1999 a way of treating patients with this symptom profile was developed in our clinic. Through a process of trial and error a frequency combination was found that relieved intense local neck pain from a cervical disc bulge within minutes. When this frequency was polarized positive from the neck to the feet it removed the full body pain from fibromyalgia associated with cervical trauma. The new technique eliminated pain in one CTF patient after months of unsuccessful treatment in the clinic for myofascial pain. By the end of 1999 25 of these patients had been treated, by April of 2001 an additional 29 had been treated, and between April 2001 and June 2002 an additional 33 were seen for a total of 87 patients. Average chronicity in this group is 10 years with a range of 1–50 years.



**Figure 9.6** Sensory diagram typical of cervical trauma-induced fibromyalgia. These patients have specific dermatomal hyperesthesia in addition to the allodynia common to all fibromyalgia patients.

These patients were unresponsive to previous medical treatments, to functional and natural medicine approaches, and to microcurrent treatment of myofascial tissue. Indeed, in some cases their pain was worsened by microcurrent myofascial treatment. The pain is not easily managed even with narcotic medication. One patient had been treated for over a year in our clinic and had had cervical disc surgery, lumbar disc surgery and a shoulder repair following an automobile accident. Her case is one that illustrates and supports the deafferentation hypothesis. Repairing the cervical disc did not change her full body pain. She moved away from Portland in 1998 and returned for one treatment in October 1999 after we had developed this treatment protocol. She left the treatment room pain free for the first time in 2 years. She had some arthritis pain but the debilitating neuropathic pain was gone following this treatment protocol. We hypothesize that the damage to the anterolateral pathways created by the disc injury perpetuated her pain and persisted despite repair of the disc. We must also hypothesize that the microcurrent and the frequencies somehow change the conductivity and function of the injured cord areas and restore proper function (Fig. 9.7).

A standard two-channel microcurrent instrument with two digit frequency settings and a three-place multiplier was used. The frequencies used in the early cases were 40Hz on channel A, thought to reduce inflammation, and 10Hz on channel B, thought to address the spinal cord. Subsequent refinements in the treatment protocol include treating with the frequencies thought to address 'chronic inflammation' in the 'cord' and 'midbrain' and 'fibrosis' in the 'cord' (the tissues and conditions are given in quotation marks because it will not be certain that a particular tissue is being treated or a particular condition is being resolved until more basic research has been undertaken to document these effects). The addition of these new frequencies has improved outcomes in the more recent patient groups treated. Based on current clinical experience it seems that the current must be used polarized positive at the upper cervical spine and negative at the sacrum or feet, depending on the extent of the patient's pain. The cervical contact wraps around the neck to the exiting nerve roots. The clinical protocol used involves wrapping the graphite conducting gloves in a small warm wet towel which is then wrapped around the neck to provide this current distribution. However any current distribution method that encircles the cord would presumably be as effective.



**Figure 9.7** The patient is usually treated prone but may be treated supine if that is more comfortable. The graphite glove from the channel polarized positive is wrapped in a warm wet towel wrapped around the neck. The graphite glove from the channel polarized negatively is wrapped in a warm wet towel wrapped around the feet.

The treatment starts to reduce the subjective pain within 10 minutes, beginning with the feet and moving cephalad until just the arm and hand pain remain. The time required to reduce the pain from incoming average of 7.3 (range 5–10/10) to the ending average of 1.3 (range 0–4/10) is about 90 minutes on the first treatment and about 60 minutes on subsequent treatments. In general, the time required to eliminate the pain becomes shorter at each subsequent treatment session. The arm and hand pain is treated with 396Hz on channel B, thought to address peripheral nerves, and 40Hz on channel A using current polarized positive at the neck and negative at the hand.

All patients with 'simple' fibromyalgia associated with cervical trauma, regardless of chronicity, experienced relief with these frequency combinations. No other frequency combinations were effective. This frequency combination is not effective in any other type of pain therefore patient selection is important. Patients were chosen on the basis of their symptom description, pain diagram, physical examination findings and a history of cervical trauma as the mechanism of onset of their chronic pain. The cervical injuries were from motor vehicle accidents, falls, lifting injuries and following surgery (presumably due to hyperextension of the neck during intubation for anaesthesia).

## Clinical controls

These treatments have been carried out in an active clinical practice funded by patients and their insurance companies and blinded placebo controlled trials are not possible in this setting. However, every effort was made to ensure that the clinical effects were produced by the treatment and not some other factor. The patients are always positioned so they cannot see the instrument. Sham treatments with the machine turned off were performed in about one quarter of the cases, at first by accident and later as an intentional control during the first portion of the



treatment to evaluate placebo effect. The sham treatment is easy to do because the current is subsensory. Sham treatment did not produce pain reduction or tissue softening in any patient. The patients were always blinded to the frequency used and to the current polarization. Four frequency combinations and both biphasic and polarized current were applied in every case. The treatments are done with the gloves wrapped around the neck and feet in a warm wet towel to remove the effect of the operator's field. In most cases the clinician leaves the room while the current is running to minimize the effect of intention and placebo. The results consistently demonstrate that placebo has a minimal effect and that the treatment protocol is producing the observed effect.

## Complete treatment protocol

Patients were treated in our clinic twice a week for 4–6 weeks with microcurrent. Massage and manipulation were used as needed. When treatment in the clinic was effective in reducing the patient's pain, the patient was sent home with a small pocket sized microcurrent unit made by Rehabicare (New Brighton, Minnesota, 800343–0488). This unit has the capacity to provide polarized current on two different channels and a number of different frequencies, including two of those we find useful. When the patients use a home microcurrent unit they must use 2x3 inch pads touching over the spinous processes and wrapping the neck laterally to the exiting nerve roots. Smaller pads do not have the desired effect. The patients are instructed to use the home unit often enough to keep their pain below a 3/10 VAS. Most patients use the home unit 6–10 hours a day; many sleep with it on so they awaken pain free. For some reason the home unit is not quite as effective as the clinic unit. It uses a sine wave instead of a square wave and lacks the high frequency harmonics that seem to be useful in sedating nerves and this may contribute to its reduced efficacy. Most patients found that after an initial period of about a month wearing the home unit 10–20 hours a day, they could reduce wearing time to 4 hours every day or every other day to maintain the desired level of comfort. The wearing time for the home unit was further reduced to once or twice a week in most patients after 2 months use. Most patients continue to use the home unit but a few patients have been able to discontinue the use of the home unit altogether.

Reducing or eliminating the pain has proved to be surprisingly easy but returning the patient to full health is a more challenging multidisciplinary effort involving exercises to stabilize the cervical spine, treatment of myofascial pain, injections for cervical or lumbar facet generated pain or persistent nerve root pain, medication withdrawal, nutritional and psychological support and physical rehabilitation.

Patients are referred to their medical provider for medication management and withdrawal, to a physical therapy clinic that specializes in spinal stabilization and exercise rehabilitation, and to a medical physiatrist certified in spinal injection procedures. Our clinic directed, monitored and coordinated care and provided treatment for nerve pain, cord mediated pain, myofascial pain and the recovery of various systems impacted by fibromyalgia such as digestion and adrenal function using microcurrent, non-prescription therapeutics and nutritional support. We also recommended the common sense lifestyle changes (e.g. increasing water consumption, dietary modification, good sleep hygiene and gentle aerobic exercise) common to most effective fibromyalgia treatment protocols.

Once the pain is eliminated or markedly reduced, the neuroendocrine system seems to right itself and the neurohormonal and digestive disturbances common to fibromyalgia improve. Neeck & Reidel (1999) proposed – and it seems a reasonable hypothesis – that the pain itself serves as a chronic stressor elevating CRH in the hypothalamus. CRH in turn modifies levels of luteinizing hormone-releasing hormone (LHRH), thyroid-stimulating hormone (TSH) and growth hormone (GH) centrally contributing to disruptions in gonadal hormones, particularly progesterone, thyroid hormone and thyroid receptor sensitivity and growth hormone levels. Other than providing adrenal support with microcurrent, nutritional supplements and herbs and removing the pain as a perpetuating factor, no direct treatment for neuroendocrine disruption is provided (Box 9.2).

Nutritional support included a low dose mixed antioxidant combination product (see Box 9.2) that provided not only antioxidants but the nutritional substrates necessary for liver detoxification pathway function in every patient. Aside from this constant, the treatment protocols were customized for each patient's particular symptom constellation. Irritable bowel syndrome was treated with avoidance of potentially allergenic foods such as wheat and milk, nutritional supplements such as L-glutamine, herbs in combinations thought to be useful for intestinal repair and support, and products that provided adequate doses of appropriate gut bacteria and nutrients to support their proliferation. Many of these patients were adrenal depleted and adrenal support products containing nutrients such as pantothenic acid, B<sub>6</sub> and vitamin C and various herbs supplied in proprietary blends helped with energy and adrenal recovery.

### Box 9.2 Ingredients in a combination low dose mixed antioxidant nutritional supplement

Vitamin A (as mixed carotenoids)	7500 IU
Vitamin C (ascorbic acid)	300 mg
Vitamin E (d-alpha tocopherol acetate and mixed tocopherols)	90 IU
Zinc (zinc gluconate)	15 mg
Selenium	75 mcg
Coenzyme Q10	3 mg
Potassium Sorbate	15 mg

Glutathione	30 mg
L-methionine	105 mg
Taurine	105 mg
n-Acetyl- Cysteine	105 mg
Superoxide dismutase	90 mcg
Catalase	90 mcg

There are microcurrent frequencies thought to address various organs and tissues in the body. Microcurrent was used with these frequencies to supply electrons at physiologic amperage. It is presumed that this increases ATP production in these organs and supplies whatever support the frequencies might have for the tissues treated. Research data are not available regarding specific effects when treating the adrenals, liver or intestines but the clinical response has been consistently positive: patients appear to recover more quickly with the use of microcurrent than without it. Improvements in intestinal palpatory pain, skin tone, affect and energy level are commonly observed, often by the end of a 20-minute treatment. Residual benefits vary and micro-current is seldom sufficient to produce lasting improvement without the use of nutritional support and lifestyle changes. However, in many cases nutritional support and lifestyle changes without the use of microcurrent have failed to produce improvement, or have resulted in only modest degrees of improvement. The combination of nutritional support and microcurrent appears to produce noticeable improvement in function, usually within 2–4 weeks. Such a rapid rate of improvement helps to create a positive expectation in the patient and contributes to both adherence and recovery.

Eliminating the deep bothersome nerve pain is the major feature of the treatment and recovery process. One patient who had developed fibromyalgia 7 years previously, following surgery for Crohn's disease, had been treated for 18 months by a medical internist skilled in functional nutritional treatment. She stated that she felt better but her pain was unchanged. Her internist referred her for treatment and her pain was eliminated at the first visit. She was treated twice in our clinic, used the home unit for 1 month, and saw a physical therapist for cervical stabilization exercises. She was pain free and fully recovered in 6 weeks and remains so 1 year later. It seems highly probable that neither approach would have worked by itself as quickly as the combination of both treatments.

## Side-effects and adverse reactions

In all but one patient, there have been no side-effects except for skin irritation from the continuous use of microcurrent adhesive pads. Some of these patients have been using the current in the fashion described above daily for 12 months, with no ill effects. One patient had headaches following treatment and treatment was abandoned. Some patients get small skin sores from the constant flow of polarized current concentrated at the outflow on the adhesive gel conductive pad. This side-effect is the most common and most bothersome. We have used skin creams and larger pads to disperse the current to minimize this effect and we are developing conductive fabric collars that may eliminate it.

Some patients do not tolerate treatment. In the early stages of the development of this treatment protocol approximately 10% of patients (five out of the first 54) had an increase in midscapular pain, followed by a headache, within 3 minutes of the application of microcurrent. Treatment was terminated and the pain returned to pre-treatment levels within 24 hours. All of the patients who did not tolerate treatment had some degree of cord compression or frank stenosis and very hyperactive reflexes. The increase in midscapular pain follows a discogenic pain pattern described by Cloward (1959) and we hypothesize that cervical cord compression from degenerated discs restricted the flow of spinal fluid causing discogenic pain and eventually headache. Improvement in the treatment technique has reduced this percentage to 5%, but these patients remain very challenging. Some eventually require surgery for the stenosis.

Of the first 54 patients treated 31 recovered from fibromyalgia after approximately 4 months of treatment. Two patients relapsed when they discontinued use of the home unit. The patients were considered to have recovered when their pain level was consistently below 3/10 or they had less than 11/18 tender points, tender to less than 4kg/in<sup>2</sup>, or when they discontinued care and self-reported that they had recovered. This recovery percentage has been maintained in the subsequent patients treated with this programme.

Not all patients could make the psychological leap from chronic pain to pain free by the end of the first 90 minute treatment. Of the first 54 patients treated five did not tolerate treatment and 18 discontinued treatment in spite of their ability to become pain free. The patients who discontinued treatment experienced a decrease in pain from an average of 7.5 to 1.3 by the end of the first treatment, which was not significantly different from the group that recovered with an almost identical drop in pain in the two groups. The vast majority of patients who discontinued treatment did so very early, usually within the first 4–6 weeks, precluding any significant chance of full recovery. The psychological component of their withdrawal from treatment would seem to represent at least as great a feature as the financial and insurance coverage reasons cited by these patients as their reason for discontinuing care.

Reversing the widely held belief that fibromyalgia is incurable and that the individual must learn to 'live with the pain' has proved to be the most challenging aspect of treatment. The most delicate period of adjustment occurs during the first 4 weeks when the patients must reverse their self-image as a dependent chronic pain patient, and

move ahead into the unknown territory of recovery. Those with determination, a strong ego sense and good personal support systems seem to be most successful in making this transition.

## Case report

Recovery has been achieved in enough cases to provide hope and therapeutic direction for this difficult condition. The typical patient with an optimal response was a 49-year-old woman referred to our clinic by her pain management group. She had had fibromyalgia for 18 years following an automobile accident. Her presenting symptoms included burning, aching, stabbing, shooting pain in the neck and midscapular area, arms, back, legs and gluteals, hands and feet rated as 7–8/10 while on narcotics and varying between a 4/10 and 8/10. She also had asthma, allergies, acne, irritable bowel syndrome and required medication for sleep. She had a positive attitude, good self-esteem, a strong healthy support system at home and insurance coverage for the treatment programme.

The patient had 20 treatment sessions between 8 December 1999 and 15 March 2000. She had the typical treatment including the in-office microcurrent protocol described above, daily use of the home unit, massage at each visit, microcurrent trigger point therapy (described later in this chapter), manipulation as needed, an epidural at C5 and facet injections in the lumbar and cervical spine, physical therapy exercises to stabilize the neck and low back, and exercise such as swimming for general reconditioning. She took nutritional supplements including mixed low dose antioxidants, magnesium and malic acid, oil based vitamin A, and a herbal fibre-rich probiotic supplement for the irritable bowel. Once she was off antidepressants she took 5-hydroxytryptophan (5-HTP) for 4 weeks to increase serotonin levels and help her sleep.

On the first day of treatment the patient had 14/18 tender points tender to pressure of less than 4lb/in<sup>2</sup>. On 12 January 2000 she had 11/18, and on 8 February 2000 7/18 tender points. By 12 May 2000 she had 4/18 tender points. She was off narcotics and muscle relaxants but still took some pain medication occasionally. She was sleeping well with no sleep medication or 5-HTP. Cervical range of motion increased by 40%, and lumbar range of motion was full and pain free. The IBS and acne had resolved and the asthma was not active. She recovered fully in 5 months, was followed for an additional 2 months before she moved out of the area and at a 1 year follow-up she was still doing well.

## Documenting objective changes

As more patients were treated and the rapid pain reductions and apparently successful outcomes were observed, a method by which to measure objective changes in serum chemistry occurring during treatment became available. It was hoped that this might explain the rapid reductions in subjective pain. Terry Phillips, PhD, an immunochemist in Bethesda Maryland, offered to do sample analysis on finger stick blood samples to identify and quantify changes of the inflammatory cytokines, interleukin-1, 6, 8, and TNF alpha and cortisol and the neuropeptides, substance P, neuropeptide Y, beta endorphins and serotonin. Over the next year samples were taken on six patients. The following patient and her serum data are representative of that group.

The patient was a 29-year-old woman who had had a lifting injury at work in 1992, 6 years prior to treatment in our clinic for myofascial pain in the neck, arms and hands in 1998. The myofascial pain was eventually found to be secondary to disc injuries at C5–6 and C6–7 and she had a cervical discectomy and fusion at these levels in 1999. Following the surgery the pain generalized to the lower extremity. By the time she was seen in our clinic for treatment of fibromyalgia on 11 March 2000 she rated her pain as 7/10, varying between a 4 and an 8/10, and described aching and burning in the neck, arms, shoulders, hands and feet with burning pain up the legs into the gluteals. She had 14 of 18 tender points tender to less than 4lb/in<sup>2</sup> pressure and required medication to sleep. She was taking prescribed anti-inflammatory and antidepressant medication. She was treated with the protocols described above and her pain was reduced to 0/10 in the first 90-minute treatment. The response was similar in the two subsequent treatments. Her pain was reduced from an average of 7/10 to 0/10 at the first visit. The pain remained reduced for up to 24 hours following treatment.

The residual pain relief from a single treatment varied from patient to patient lasting from 4 hours up to 4 days, with some improvements lasting as long as 2 weeks.

Finger stick blood samples were taken before, during, and at the end of each treatment, air dried overnight and sent to Dr Phillips at his Bethesda laboratory. The first sample was taken before treatment and the subsequent samples were taken as the pain dropped, or frequencies were changed. On each treatment date 90 minutes elapsed between the first and final sample. The changes in serum chemistry at the three treatments are noted in [Table 9.1](#). The changes for the cytokines, substance P, endorphins and cortisol are linear and correlate directly with pain score reduction.

The serotonin response is intriguing and follows a different pattern from that of the other variables. Serotonin dropped while the pain was coming down and continued to drop until the pain reached 0/10. When the pain became 0/10 we changed to two frequencies thought to reduce 'nervous tension' and 'emotional tension'. When these

frequencies were used the patients reported feeling very relaxed and experienced an almost hypnotic state. This was a very different effect than the profound relaxation produced during the pain relief phase, presumably produced by the increases in endorphins and cortisol. In every case serotonin reversed its downward trend and rose, in this case by 71%, when these frequencies were used. In one case serotonin increased from 155 to 315 – an increase of 103%. These frequencies produce this relaxation effect routinely and no other frequencies have the same effect, even when both the patient and the provider are blinded to the frequency being used. The effect is more profound when the level of emotional tension and nervous tension are high and more subtle when they are low. The effect lasts for approximately 1 hour although patients report feeling relaxed for up to 24 hours. No hypothesis has been developed to explain how or why the effect occurs, or why serotonin increases with the use of these frequencies, but the effect is consistent and suggests an area ripe for further research.

**Table 9.1** Serum sample data from a cervical trauma patient

Sample	Date (2000)	IL-1	IL-6	IL-8	TNF-a	IFNg	SP	VIP	b- endorphin	Cortisol	Serotonin
M1	11 May	392.8	204.3	59.9	299.1	97.2	132.6	8.5	5.2	15.5	285.6
M2	11 May	288.5	200.8	47.6	265.7	99.8	127.5	10.2	7.1	12.6	309.2
M3	11 May	103.2	121.7	21.3	96.5	73.7	82.4	32.9	21.4	33.7	202.1
M4	11 May	52.6	33.9	11.4	43.4	32.6	38.2	48.4	69.1	78.3	169.5
M5	11 May	21.4	15.6	4.8	20.6	11.4	10.5	69.9	88.3	169.9	289.6
M1	14 May	218.7	165.9	45.7	205.6	75.9	99.6	4.9	9.4	12.9	250.0
M2	14 May	113.2	87.3	21.6	151.8	44.7	102.5	26.3	36.7	61.8	203.7
M3	14 May	45.6	40.7	5.8	33.3	26.5	41.7	39.1	89.5	149.3	366.2
M1	17 May	145.9	100.5	42.6	114.2	80.6	144.6	12.5	11.6	11.8	322.4
M2	17 May	61.5	47.2	10.4	71.9	39.3	55.7	28.4	88.6	78.9	259.3
M3	17 May	10.6	11.6	5.1	22.1	5.9	9.4	71.8	115.9	182.6	410.6
Normal		0–	0–	0–	0–	0–	0–	0–	0–	5–25mg/	100–
Range		25pg/ml	25pg/ml	25pg/ml	25pg/ml	25pg/ml	30pg/ml	20pg/ml	35pg/ml	ml	300ng/ml

IL-1: interleukin-1, IL-6: interleukin-6, IL-8: interleukin-8, TNF-a: tumour necrosis factor alpha, IFNg: interferon gamma, SP: substance P, VIP: vasoactive intestinal peptide, b-endorphin: beta endorphin.

As it happens, this cervical trauma patient did not persist in treatment beyond the first five sessions due to financial constraints, did not acquire a home unit, would not allow herself to be treated at no cost and she has not recovered as far as we know.

It is apparent that the inflammatory cytokines were moderately elevated in this patient, as they were in all six patients sampled, who had fibromyalgia associated with cervical trauma. One patient who presented with a diagnosis of fibromyalgia but who actually had simple upper extremity and lumbar myofascial pain had no elevations in cytokines and her levels are shown in [Table 9.2](#). She did not respond to the CTF protocol but her pain was eliminated with the treatment protocol for myofascial pain and trigger points (see below). Her blood samples were taken before and at the end of treatment. Her pain was 0/10 at the end of this treatment and she recovered from her myofascial pain after six treatments, use of a magnesium malate supplement and reconditioning.

## TREATMENT OF MYOFASCIAL PAIN

Most fibromyalgia patients have myofascial pain or trigger points, but patients with myofascial pain syndrome do not necessarily have the full neuroendocrine profile seen in fibromyalgia. Successful resolution of myofascial pain is essential to the recovery of the fibromyalgia patients discussed above. It has been suggested that patients with simple myofascial pain may progress to fibromyalgia if left untreated. There are multiple theories as to the aetiology, physiology, pathology and perpetuating factors associated with myofascial pain syndrome and trigger points. Microcurrent treatment with specific frequencies did not evolve from any of the theoretical resources; it was a purely clinical development. Different frequency combinations were used clinically, and, purely by trial and error, treatment sequences of frequencies were developed that seemed effective. It was only later that a literature review supported the rationale for the choice and use of the frequencies we found to be effective.

Unlike traditional trigger point therapy which requires injections, or firm and often painful pressure (Travell & Simons 1983), application of microcurrent to the tissue reduces the pain and tenderness and causes the tissue to soften with minimal to no pressure. The current is used in an alternating mode with gloves placed so that the current flows through the involved muscle and its biomechanical antagonists. In order for the treatment to be effective the current must only pass through the tissue to be treated. Pressure is applied as needed to palpate the changes in the tissue as it softens. The muscles can be moved passively through a range of motion during treatment to facilitate resolution of myofibrosis but this does not seem to be essential to the process.

When the frequency is 'correct', the tissue relaxes under the therapist's fingers until that frequency has finished its



portion of the work. When the changes stop, further use of that frequency during that session is usually not productive and different frequencies must be used to produce results. Each time the correct frequency is chosen and applied there is a feeling of the tissue softening under the operator's fingers and the patient generally feels a sensation of warmth, tissue softening and pain reduction.

**Table 9.2** Myofascial pain patient

Sample	Date (2000)	IL-1	IL-6	IL-8	TNF-a	IFNg	SP	VIP	b-endorphin	Cortisol	Serotonin
J1	27 June	61.8	55.7	32.6	75.8	27.3	44.9	18.5	11.6	39.9	42.6
J2	27 June	66.4	51.7	40.3	71.8	30.3	41.6	16.4	19.5	44.7	31.8

IL-1: interleukin-1, IL-6: interleukin-6, IL-8: interleukin-8, TNF-a: tumour necrosis factor alpha, IFNg: interferon gamma, SP: substance P, VIP: vasoactive intestinal peptide, b-endorphin: beta endorphin.

The sequence of frequencies used is individualized depending on the condition of the muscles, the patient's history and physical examination and the operator's perception of the patient's response to treatment. Generally we start with a series for 'fibrosis', followed by frequencies used for 'mineral deposits' and 'allergy reaction'. It is interesting that the frequencies we find to be most effective were described as treating conditions such as fibrosis, calcium deposits and histamine release by mast cells, which coordinate with the mechanisms for myofascial dysfunction proposed by Travell and others.

There are frequencies thought to be specific for conditions such as fibrosis, scar tissue, mineral deposits, allergy reaction, chronic inflammation, toxicity and viral infection to be combined with frequencies for specific tissues such as muscles, tendons, connective tissue, arteries and nerves. We have available about 20 combinations of frequencies we use on a regular basis. We have observed, measured and palpated the effects of the frequencies, but short of dissection or biopsy, there is no way to know with certainty exactly what they are doing to any specific tissue or condition.



**Figure 9.8** The patient is positioned comfortably and the gloves used in such a way that the practitioner can treat the symptomatic jaw and cervical muscles and their antagonists simultaneously.

The response is clearly frequency specific. Time spent using an 'inappropriate' or ineffective frequency produces no change in tissue no matter how long the frequency is used. Changing to a 'correct' frequency produces the characteristic softening of the tissue in seconds. This response occurs even when the operator is unaware of the frequency being used. Trials were performed with the operator blinded to the frequencies and the tissue response was consistent, independent of the operator's expectation or knowledge.

## Treatment technique

The treatment technique made possible by use of the graphite/vinyl gloves is a real advantage in treating the sensitive musculature of the head, jaw and neck. In order to be effective the current must simply pass through the dysfunctional tissue. Compression is not essential to the process. This makes it possible, for example, to treat the suboccipital muscles by inserting one glove into the buccal area at the back of the mouth and placing the other on the suboccipital area. This intraoral technique can also be used to treat the pterygoids, digastric, omohyoid, scalenus muscles and the cervical paraspinals. The current travels from the intraoral glove through the muscles to the external glove wherever it is placed (Fig. 9.8).

Unlike injections or ischaemic compression, which can only treat small areas, this method allows treatment of entire muscles and synergist/antagonistic muscle groups at the same time during the same visit allowing a smooth return of normal biomechanical function to the painful dysfunctional region. For example, when the upper back, shoulder and posterior neck are treated it is possible to simultaneously treat the serratus anterior, the subscapularis, the levator and trapezius, the cervical paraspinals, multifidi and scalenes in one treatment session. This seems to provide a distinct advantage in the recovery of biomechanical function as well as providing pain relief (Figs 9.9, 9.10).



**Figure 9.9** Supine treatment focuses on the scalenes and cervical paraspinal muscles.

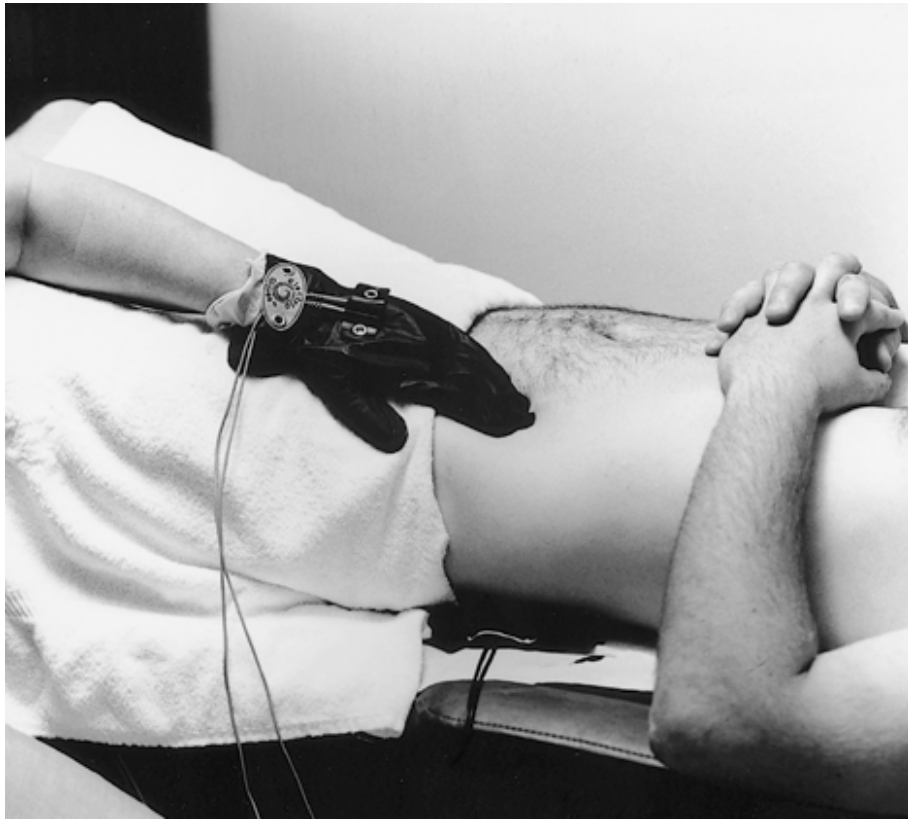


**Figure 9.10** Prone cervical myofascial treatment focuses on the cervical paraspinals, the levator and trapezius, the serratus anterior and subscapularis.

When treating low back pain and the psoas, iliacus, quadratus lumborum and lumbar paraspinals the same principles apply. The psoas is treated during the first treatment while the patient is supine with the knees and hips flexed. The gloves are positioned with the operator's gloved hand just inside the iliac crest anteriorly and the other glove, without the operator's hand, under the patient's back. This allows the current to pass through the psoas and posterior muscles simultaneously and leaves the operator one free hand to change frequencies. If there is referred pain down the thigh from the psoas (Travell & Simons 1992), the current can be polarized with the positively charged glove used on the active trigger point in the psoas and the negatively polarized glove placed on the referral area (Fig. 9.11).

The second low back treatment is done with the patient prone (Fig. 9.12). The gloves are placed on the lumbar paraspinal muscles bilaterally and the current flows from one side through to the other. One glove contact can be placed under the abdomen to treat the muscles from anterior to posterior. The patient is usually much improved after the first treatment that focuses primarily on the psoas. The second treatment seems to alleviate much of the remaining posterior pain. The subsequent treatments usually address trigger points in the gluteals, tensor fascia lata, pectineus or piriformis and maintain the tissue improvement while the patient begins reconditioning.

Unlike spray and stretch, which can be awkward to use in certain areas, microcurrent is simple and direct and allows easy access to complex muscle couples. There are no known environmental hazards associated with the use of microcurrent.



**Figure 9.11** Supine lumbar myofascial treatment focuses on the psoas.

Home stretches and exercises are prescribed within the first 2 weeks. Conditioning is gradual and gentle and designed to increase muscle oxygenation and mobility before increasing strength.

If the facet is the primary pain generator and the myofascial trigger points are thought to be secondary to or compensatory for the facet irritation, manipulation of the joint specific for facet syndrome is performed and if necessary facet injections are ordered. The prescribed exercises focus on strengthening the muscles while the facet is gapped and in traction by having the patient flex the lumbar spine and perform small movements in extension or side-bending.

If the disc has been injured and is serving as the primary pain generator and the perpetuating factor for the myofascial pain, manipulation and, if necessary, epidural injections are ordered. Exercises and postural recommendations are prescribed to keep the spine, especially the lumbar spine, in extension while the lumbar paraspinal muscles and the abdominal muscles are strengthened.

Patients are given a supplement containing magnesium glycinate 150mg, malic acid 600mg, manganese glycinate 5mg, B<sub>6</sub> 50mg and thiamine 50mg to help improve muscle function and provide enhanced nutrients for muscle metabolism. The supplement is continued during treatment and for 2 weeks after treatment is completed. If the myofascial pain begins to recur at some future time the patient is instructed to begin taking the supplement immediately and to return for treatment if the pain does not recede within 5 days. The low dose antioxidant nutritional supplement described above and in [Box 9.2](#) is also recommended to enhance antioxidant status and help the patient detoxify or process muscle metabolites released during treatment.





**Figure 9.12** Prone lumbar myofascial treatment focuses on the lumbar multifidi, paraspinals and gluteals.

## Side-effects and adverse reactions

The most common side-effect is a post-treatment reaction starting approximately 90 minutes after treatment and lasting 6–24 hours. It is presumed to be a detoxification reaction similar to that seen after massage only magnified. Symptoms include slight to moderate nausea, flu-like aching and sometimes a slight increase in pain. This reaction can usually be avoided by consumption of two quarts of water in the first 3 hours after treatment and use of the anti-oxidant supplement mentioned above that provides phase one and phase two liver detoxification pathway substrates. The reaction is less pronounced after the third or fourth visit, presumably because liver detoxification pathway enzymes increased with the increased demand.

Most patients treated for myofascial pain are observed to have a significant increase in range of motion following treatment. Some patients with a significant amount of joint degeneration, especially in the cervical spine, may also experience a temporary occurrence of radicular pain following treatment, presumed to be due to the movement of the degenerative spurs into the nerve space. This reaction can be treated with the microcurrent protocols for radicular pain. The neuropathic pain stops when the range of motion returns to normal and we hypothesize that the bone spurs are no longer moving into the nerve space. Once this reaction is gone the myofascial pain remains quiet, usually for 4–6 months. The joint degeneration perpetuates the myofascial pain, and supplements and occasional microcurrent treatments must be continued to keep these patients pain free long term.

Pain that is thought to be myofascial but which is in fact due to nerve or cord compression or irritation can actually increase when treated with the myofascial protocols. When this pain increase occurs we have found that switching immediately to the protocols for neuropathic pain reduces the pain and will eventually relieve the myofascial pain as well (Fig. 9.13).

In addition to these precautions specific to our uses of microcurrent, the general precautions and contraindications for microcurrent are observed. It is not to be used through a pregnant uterus or on patients with demand type pacemakers.



**Figure 9.13** Cervical nerve pain can be treated effectively using the frequencies thought to reduce inflammation and the frequencies thought to address the peripheral nerves. The current is polarized positively at the neck and negatively at the end of the dermatome being treated, in this case the C5 nerve root.

## Myofascial results in clinical practice

In 1996 we examined the results in 137 cases of 'simple' chronic myofascial pain in various body regions uncomplicated by disc injury, neuropathy or severe arthritides, most due to prior trauma or chronic overuse. Symptom duration ranged from 8 months to 22 years. The majority of patients had been treated by one or more prior therapies including prescription drugs, physical therapy, surgery, chiropractic, acupuncture, trigger point therapy and massage. Of those 137 patients, 128 completed treatment. Pain was reduced in 126 of those 128 from an average 5–8/10 to a 0–2/10. Two patients had pain reduced from the 5–8/10 range to 3–4/10 range. Treatment duration varied depending on the severity, complexity and chronicity of the case. Patients were told to return if the pain recurred or motion became limited. Random follow-up contacts suggest that the results have been long lasting and possibly permanent.

Further refinements in treatment techniques and frequencies resulted in improved patient response and reduced the number of treatments required to achieve resolution of the patient's symptoms. Data were retrieved from the charts of 73 patients with head, neck, face or low back pain resulting from chronic myofascial complaints seen between January and June 1997. We defined chronic as pain lasting longer than 90 days after the precipitating trauma. Most of the patients were referred to the clinic by a medical physician, chiropractor, naturopathic physician or another patient.

**Table 9.3** shows the outcomes of treatment of these patients. The results since this early data sample have remained consistent, and have even improved as both assessment and treatment techniques have been refined. Simple myofascial pain regardless of chronicity resolves quickly and usually permanently with myofascial treatment using frequency specific microcurrent, nutritional support and gentle specific rehabilitation exercising. Patients who do not respond in the expected fashion within 4 weeks are now treated with protocols for neuropathic pain and discogenic, facet generated or metabolic perpetuating factors developed since 1999. Referrals for spinal stabilization exercises, epidural and facet injections are made within 4 weeks if myofascial treatment does not produce lasting improvement. This shift in treatment programme has improved our outcomes in recent years. Similar outcomes are reported anecdotally by practitioners using this technique in clinics in the USA, Canada and Australia. Formal data collection and outcomes assessment is planned for the near future to evaluate the effectiveness in a more scientific fashion.

**Table 9.3** Outcomes in the early treatment of myofascial pain (Treatment of chronic myofascial pain: presented at the American Back Society Annual Meeting in San Francisco, 1997)

*Simple myofascial pain: averaged outcomes in 50 cases of head, neck and face pain*

Patients	Chronicity	Failed other TX	No. treatments	No. weeks	VAS start	VAS end
50	4.7 years Range (1–28)	88%	11.2	7.9	6.8/10	1.5/10
<i>Simple myofascial pain: averaged outcomes in 23 cases of chronic low back pain</i>						
Patients	Chronicity	Failed other TX	No. treatments	No. weeks	VAS start	VAS end
23	8.4 years Range (1–20)	87%	5.7	5.7	6.8/10	1.6/10

## CONCLUSION

Microcurrent therapy has improved outcomes in the treatment of myofascial pain and fibromyalgia leading to successful and complete resolution of symptoms in many cases. Microcurrent is not sufficient to produce these effects by itself but rather forms a necessary adjunct to other therapies and strategies, which by themselves are helpful but not sufficient to produce resolution in most cases. Even though complete permanent resolution cannot be achieved in every case, these outcomes in the treatment of fibromyalgia suggest that the condition is indeed curable in many patients.

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